

**Amendments to the Claims:**

This listing of claims will replace all prior versions and listings of claims in the application:

1 - 32. Canceled.

33. (New) A method for the selection, identification or characterization of compounds which can modulate reverse cholesterol transport, which comprises:

- a. contacting a test compound with a nucleic acid construct comprising, as the only LRH-1 response element, at least one copy of the LRH-1 response element of the promoter of the human apolipoprotein AI gene containing the following sequence (SEQ ID NO: 1): 5'-CTGATCCTTGAAC-3', and
- b. determining the possible binding of said test compound to the response element.

34. (New) The method according to claim 33, wherein the contact is carried out in the presence of the exogenous LRH-1 receptor or a functional equivalent thereof, and in that one determines the possible binding of said test compound to the LRH-1 response element and/or to the complex formed by the binding of LRH-1 to its response element.

35. (New) A method for the selection, identification or characterization of compounds which can modulate reverse cholesterol transport, which comprises :

- a. contacting a test compound with a host cell containing a reporter gene expression cassette, said cassette comprising a reporter gene placed under the control of a promoter comprising, as the only LRH-1 response element, at least one copy of the LRH-1 response element of the promoter of the human apolipoprotein AI gene containing the following sequence (SEQ ID NO : 1): 5'-CTGATCCTTGAAC-3', and
- b. determining the effect of the presence of the test compound on the binding of LRH-1 to the response element or on the expression of the reporter gene.

36. (New) The method according to claim 35, wherein the host cell comprises an exogenous LRH-1 receptor or a functional equivalent thereof.

37. (New) The method according to claim 35, wherein the host cell comprises a ligand of LRH-1.

38. (New) The method according to claim 35 comprising determining the level of expression of the reporter gene in the presence of the test compound and in the absence of said compound, an increase or a decrease in the level of reporter gene expression indicating the ability of the test compound to modulate reverse cholesterol transport.

39. (New) The method according to claim 35, wherein the host cell is a mammalian cell.

40. (New) The method according to claim 35, wherein the host cell is a human cell.

41. (New) The method according to claim 35, wherein the reporter gene is a gene coding for a product whose activity or presence in biological extracts can be measured, in particular one of the genes coding for luciferase, secreted alkaline phosphatase, galactosidase or lactamase.

42. (New) The method according to claim 35, wherein the promoter is selected in the group consisting of the HSV-TK promoter, the CMV immediate early promoter, the PGK promoter, the promoter of the gene coding for human apolipoprotein AI and the SV40 promoter.

43. (New) The method according to claim 33, wherein one or more compounds are tested, as a mixture or separately.

44. (New) The method according to claim 33, wherein the test compound is a combinatorial library.

45. (New) The method according to claim 33, wherein the test compound is a clone or a library of nucleic acid clones coding for one or more DNA-binding polypeptide(s).

46. (New) The method according to claim 33, wherein contact is carried out in a multiwell plate.

47. (New) The method according to claim 33, wherein additionally comprising a comparison of the possible effects determined by said method with the possible effects determined by a method carried out in the same conditions but with a nucleic acid construct containing at least one mutated copy of the LRH-1 response element of the promoter of the human apolipoprotein AI gene, containing the following sequence (SEQ ID NO : 1): 5'-CTGATCCTTGAAC-3', said mutant copy essentially being unable to bind the LRH-1 receptor.

48. (New) The method according to claim 33, for the selection, identification or characterization of compounds which can increase reverse cholesterol transport.

49. (New) The method according to claim 33, for the selection, identification or characterization of compounds which can modulate the activity of HDL.

50. (New) The method according to claim 33, for the selection, identification or characterization of compounds which can modulate the expression of apolipoprotein AI.

51. (New) A method for the modulation of reverse cholesterol transport, comprising administering to a subject in need thereof a compound which can modulate the binding of LRH-1 and/or its cofactors to the response element of the promoter of the human apolipoprotein gene or a functional variant thereof.

52. (New) A method for the increase of reverse cholesterol transport, comprising administering to a subject in need thereof a compound increasing the binding of LRH-1 and/or its cofactors to the sequence SEQ ID NO : 1 or a functional variant thereof.

53. (New) A method for the modulation of the activity of HDL, comprising administering to a subject in need thereof a compound modulating the binding of LRH-1 and/or its cofactors to the sequence SEQ ID NO : 1 or a functional variant thereof.

54. (New) A method for the increase of the activity of HDL, comprising administering to a subject in need thereof a compound increasing the binding of LRH-1 and/or its cofactors to the sequence SEQ ID NO : 1 or a functional variant thereof.

55. (New) A method for modulating the expression of ApoAI, comprising administering to a subject in need thereof a compound modulating the binding of LRH-1 and/or its cofactors to the sequence SEQ ID NO : 1 or a functional variant thereof.
56. (New) A method for the modulation of reverse cholesterol transport, comprising administering to a subject in need thereof a compound increasing the effect of LRH-1 and/or its cofactors on the transcription of the human apolipoprotein AI gene.
57. (New) The method according to claim 51, characterized in that the compound is a nuclear factor or a cofactor.
58. (New) The method according to claim 51, characterized in that the compound is a clone expressing one or more DNA-binding polypeptide(s).
59. (New) The method according to claim 51, characterized in that the compound is a compound which is selected, identified or characterized by:
- a. contacting a test compound with a nucleic acid construct comprising, as the only LRH-1 response element, at least one copy of the LRH-1 response element of the promoter of the human apolipoprotein AI gene containing the following sequence (SEQ ID NO: 1): 5'-CTGATCCTTGAAC-3', and
  - b. determining the possible binding of said test compound to the response element.
60. (New) A nucleic acid fragment characterized by the following sequence (SEQ ID NO : 1): 5'-CTGATCCTTGAAC-3'.
61. (New) An expression cassette comprising at least one copy of the nucleic acid fragment as defined in claim 60, and a promoter, selected from among the CMV immediate early promoter and the PGK promoter, associated with a reporter gene placed under the control of said promoter.
62. (New) A method for *in vitro* screening of compounds which can modulate the activity of HDL, wherein the cassette as defined in claim 61 is used.

63. (New) A pharmaceutical composition, comprising a compound which is selected, identified or characterized according to claim 33.